COLLOID AND METHYLPREDNISOLONE THERAPY AS ALTERNATIVE MANAGEMENT OF DHF

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ABSTRACT

There are three phase in progresivity of dengue infection which are afferent phase, efferent phase and efector phase. In dengue infection, it has been found endothelial cells leakage, the proinflammatory cytokine level alteration and other mediator followed by the plasma migration and it has the potency to become dengue shock. The main clinical manifestation are fever and bleeding. The aim of this research is to prove the influence of colloid and methylprednisolone theraphy to inhibit the endothelial cells leakage through the alteration of IL-1β, TNF-α and PLA-2 level. This research has been involved 36 dengue patients in Tropical Infection Division, Departement of Internal Medicine Medical Faculty Airlangga University - Dr. Soetomo Hospital Surabaya and 36 control group. We did the examination of cytokine level (IL-1β, TNF-α, PLA-2) on blood sample with ELISA method. The dose methylprednisolone 125 mg twice a day and colloid (MW 40 kDa) 500cc per day for 3 days has been given to the patient whose fulfilled the inclusion criteria. We found temperature change to normal level after intervention. In conclusion, colloid and methylprednisolone therapy is effective to accelerade the temperature, IL-1β, TNF-α, and PLA-2 level declination, accelerate the endothelial cells sealing and plasma migration in dengue infective patient.

Key words: *DHF, Colloid, Methylprednisolone, proinflammatory cytokine*

ABSTRAK

Progresivitas infeksi dengue terjadi melalui tiga fase yaitu fase aferen, eferen dan efektor. Pada infeksi demam berdarah dengue terjadi kebocoran endothelial cells, perubahan kadar sitokin proinflamantori serta mediator lain diikuti oleh perpindahan plasma dan berpotensi menjadi syok dengue. Manifestasi klinis menonjol adalah demam disertai perdarahan. Tujuan penelitian adalah membuktikan pengaruh pemberian koloid dan metilprednisolon untuk menghambat kebocoran endothelial cells melalui perubahan kadar IL-1 β dan TNFα serta PLA2. Penelitian ini dilakukan melibatkan 36 penderita DBD yang dirawat di Divisi Penyakit Tropik Infeksi, Departemen Penyakit Dalam Fakultas Kedokteran Universitas Airlangga - RSU Dr. Soetomo Surabaya dan 36 kontrol orang sehat. Metode penelitian sampel darah diperiksa kadar sitokin (IL-1β, TNFα, PLA-2) dengan metode ELISA. Metilprednisolon dosis 2 kali 125 mg dan koloid (BM 40 kDa) 500cc per hari, selama 3 hari diberikan pada penderita yang memenuhi kriteria inklusi. Hasil penelitian terjadi perubahan suhu tubuh ke arah normal karena terjadi perubahan kadar IL1β, TNFα, dan PLA2 setelah pemberian koloid dan metilprednisolon. Kesimpulan, pemberian koloid dan metilprednisolon efektif mempercepat penurunan suhu, penurunan kadar IL-1β, TNFα, mencegah dan mempercepat penutupan endothelial cells serta perpindahan plasma pada penderita terinfeksi virus dengue.

Kata kunci: *DBD, Koloid, Metilprednisolon, sitokin proinflamatori*

Submit: 5 Mei 2010, Review 1: 12 Mei 2010, Review 2: 12 Mei 2010, Eligible articles: 1June 2010

INTRODUCTION

Dengue infection may range from asymptomatic to severe hemorrhagic fever and fatal dengue shock syndrome (DSS). Dengue fever is usually a nonspecific, self- limited biphasic febrile illness. A subset of patients, often with secondary infection, may progress to dengue shock syndrome in which acute fever, hemorrhagic manifestations, and marked capillary leak are prominent; the letter manifests as pleural effusions and ascites, and there is a tendency for shock to develop.

There are three phase in progresivity of dengue infection which are afferent phase, efferent phase

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and efector phase. In efferent phase, dengue virus has been influenced macrophage as target cell to produce and increase proinflammatory cytokine secretion (IL-1β, IL-6, TNF-α) and also phospholipase-2 enzyme (PLA-2). This PLA-2 enzyme could influence arachidonate acid metabolism and trigger secondary mediator formation (thromboxan-A2, leucotrien, prostacycline and prostaglandin-E2). Many mediators and enzyme could cause to capillary endothelial cells leakage, plasma migration. If this condition happened subsequently, it would stimulate the dengue shock syndrome (DSS). To prevent and handle endothelial cells leakage, we need colloid which has sealing effect and could be in vessel in long time.

The key is early recognition and understanding of the clinical problems during the different phase of the disease, leading to a rational approach to case management and a good clinical outcome. A well-managed front-line response not only reduces the number of unnecessary hospital admissions but also saves the lives of dengue patients.

Reducing dengue mortality requires an recognized process that guarantees early recognition of the disease, and its management and referral when necessary. The key component of the process is the delivery of good clinical services at all levels of health care, from primary to tertiary levels. Most dengue patients recover without requiring hospital admission while some may progress to severe disease. Simple but effective triage principles and management decisions applied at the primary and secondary care levels, where patients are first seen and evaluated, can help in indetifying those at risk of developing severe disease and needing hospital care. This should be complemented by prompt and appropriate management of severe dengue in referral centres.

Recently WHO recommendation for Management (2009) or a stepwise approach to the management base on: step one (history, physical and laboratory examination); step two (diagnosis, assessment of disease phase and severity); step three (disease notification management). Treatment is directed toward symptomatic relief, fluid therapy and management of complications. Treatment entails fluid therapy, the appropriate use of volume support crystalloid and or colloids.

Based on some phenomena above, we need to perform research to gain deeply understanding and detailed information.

METHODS

This research has been involved 36 dengue patients in Tropical Infection Division, Departement of Internal Medicine, Medical Faculty Airlangga University - Dr. Soetomo Hospital Surabaya period November 2008 to June 2009 and 36 control group. Each patient should fulfilled informed consent. Laboratory examination consist of whole blood, liver function test, renal function test, serum electrolite, blood glucose every day. The dose methylprednisolone 125 mg twice a day and colloid (MW 40 kDa) 500cc per day for 3 days has been given to the patient whose fulfilled the inclusion criteria. Cytokine level examination (IL-1β, TNF-α) and PLA-2 has been perform on day-1, day-3 after methylprednisolone and colloid intervention. Laboratory examination where perform in Clinical Pathology Departement and Institute of Tropical Disease Airlangga University. Dengue Infection diagnosis based on WHO criteria and PCR dengue typing. Research procedure has been agreed by ethical commite of Dr. Soetomo Hospital Surabaya.

This research is single blind randomized controlled trial whereas infected dengue patient has been prooved clinically and laboratories as well as WHO criteria give methylprednisolone and colloid for 3 days. Follow up of each patient has been perform until 7 days of fever period. During this period will have been perform laboratory examination especially TNF-α, IL-1β, PLA-2 before and after short-term methylprednisolone and colloid therapy. Temperature level where perform every day. The data has been analyzed by SPSS 15.

RESULTS

Characteristic of Research Subject

The distribution of patients sex: Man 22 (61,1%), Woman 14 (38,9%)

Table 1. The distribution of Age:

Age (years)	Total	
	n	%
<20	22	61.1
20–29	9	25.0
30–39	4	11.1
40–49	1	2.8
Total	36	100

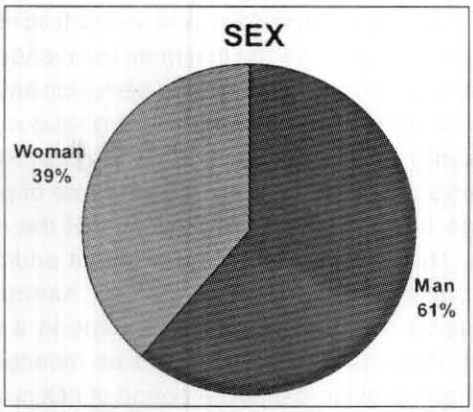

Figure-1. The proportion of subject gender. The proportion of man group is 61.1% and 38.9% for woman group.

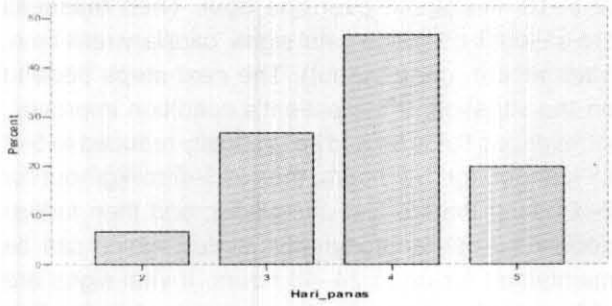

Figure-2. The day of fever proportion. The proportion of fourth day fever is 46.7%, the third day proportion is 26.7%, the fifth day of fever proportion is 20%, and the second day of fever is 6.7%.

Table 2. The distribution of Disease Severity:

Dengue infection	Total	
	N	%
Dengue Fever	8	22.2
DHF grade I	23	63.9
DHF grade II	4	11.1
DHF grade III	1	2.8
DHF grade IV	0	0
Total	36	100

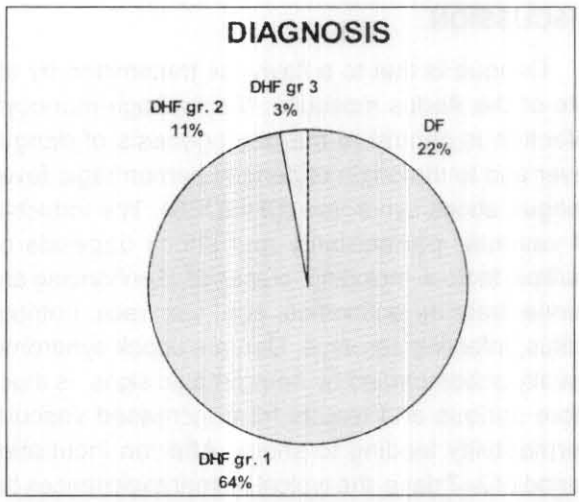

Figure-3. The proportion of diagnostic category. Subject proportion with DF 22.2%, DHF grade I is 63.9%, DHF grade II is 11.1%, DHF grade III is 2.8%.

There is no significant differences in temperature ($p = 0.082$; significant if $p < 0.050$) parameter between pre and post intervention using methylprednisolone (Medixon®) and colloid (Haemacell®).

Table 4. Table of Cytokine and PLA-2 Enzyme Level Alteration in Methylprednisolone and Colloid Group Test Statistics(c)

	PLA2_4 - PLA2_1	TNFa_4 - TNFa_1	IL1b_4 - IL1b_1
Z	-3.296(a)	-3.180(b)	-3.233(a)
Asymp. Sig. (2-tailed)	.001	.001	.001

a Based on negative ranks.
b Based on positive ranks.
c Wilcoxon Signed Ranks Test

There are significant differences between pre and post intervention using methylprednisolone (Medixon®) and colloid (Haemacell®) ($p = 0.001$ for PLA-2, $p = 0.001$ for TNF-α, and $p = 0.001$ for IL-1β, significant if $p < 0.050$).

Table 3. Paired T-Test of Colloid and Methylprednisolone Group Paired Samples Test

		Paired Differences					T	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Temperature_ax_0 – Temperature_ax_4	.93333	.49329	.28480	-.29206	2.15873	3.277	2	.082

DISCUSSION

Dengue is due to a flavivirus transmitted by the bite of the *Aedes* mosquito. Macrophage-monocyte infection is central to the pathogenesis of dengue fever and to the origin of dengue hemorrhagic fever/ dengue shock syndrome (DHF/DSS). The induction of vascular permeability and shock depends on multiple factors, including: presence of enhancing and nonneutralizing antibodies, age, sex, race, nutrition status, infecting serotype. Dengue shock syndrome, usually accompanied by hemorrhagic signs, is much more serious and results from increased vascular permeability leading to shock. After an incubation period of 2–7 days, the typical patient experiences the sudden onset of fever, headache, retroorbital pain, and back pain along with the severe myalgia. The illness may last a week, with additional symptoms usually including anorexia, nausea or vomiting, marked cutaneous hypersensitivity, and near the time of defervescence, a maculopapular rash beginning on the trunk and spreading to the extremities and the face. The maculopapular rash that often develops in dengue fever may also appear in DHF/DSS. Epistaxis and scattered petechiae are often noted in uncomplicated dengue, and preexisting gastrointestinal lesions may bleed during the acute illness. In more severe cases, frank shock is apparent, with low pulse pressure, cyanosis, hepatomegaly, pleural effusions, ascites, and in some cases severe ecchymoses and gastrointestinal bleeding.

Treatment, no specific antiviral therapy. Treatment is directed toward symptomatic relief, fluid therapy and management of complications.

WHO guidelines for treatment (2009), for in hospital management, if the patient has dengue with warming signs, the action plan should be as follows: obtain a reference haematocrit before fluid therapy. Give only isotonic solutions such as 0.9% saline, Ringer's lactate, Ringer's acetate. Start with 5–7 ml/ kg/hour for 1–2 hours, than reduce to 3–5 ml/kg/hour for 2–4 hours, and then reduce to 2–3 ml/kg/hour or less according to clinical response. Reassesses the clinical status and repeat the haematocrit. If the haematocrit remains the same or rises only minimally, continue with same rate (2–3 ml/kg/hour) for another 2–4 hours. If the vital signs are worsening and haematocrit is rising rapidly, increase the rate to 5–10 ml/kg/hour for 1–2 hours. Reassess the clinical status, repeat the haematocrit and review fluid infusion rates accordingly. Give the minimum intravenous fluid volume required to maintain good perfusion and urine output of about 0.5 ml/kg/hour. Intravenous fluids are usually needed for only 24–48 hours. Reduce intravenous fluids gradually when the rate of plasma leakage decreases towards the end of the critical phase. This is indicated by urine output and/or oral fluid intake that is/are adequate, or haematocrit decreasing below the baseline volume in a stable patient. Patients with signs should be monitored by health care providers until the period of risk is over. A detailed fluid balance should be maintained.

The action plan for treating patients with **compensated shock** is as follows: start intravenous fluid resuscitation with isotonic crystalloid solutions at 5–10 ml/kg/hour over one hour. Then reassess the patient's condition (vital signs, capillary refill time, haematocrit, urine output). The next steps depend on the situation. If the patient's condition improves, intravenous fluids should be gradually reduced to 5–7 ml/kg/hour, for 1–2 hours, then to 3–5 ml/kg/hour for 2–4 hours, then to 2–3 ml/kg/hour, and then further depending on haemodynamic status, which can be maintained for up to 24–48 hours. If vital signs are still unstable, check the haematocrit after the first bolus. If the haematocrit increases or is still high (>50%), repeat a second bolus of crystalloid solution at 10–20 ml/kg/hour for one hour. After this second bolus, if there is improvement, reduce the rate to 7–10 ml/kg/hour for 1–2 hours, and then continue to reduce as above. If haematocrit decreases compared to the initial reference haematocrit (<40% adult females, 45% in adult males), this indicates bleeding and the need to cross-match and transfuse blood as soon as possible. Further boluses of crystalloid or colloid solutions may need to be given during the next 24–28 hours.

Patients with **hypotensive shock** should be managed more vigorously. The action plan for treating patients with hypotensive shock is as follows: initiate intravenous fluid resuscitation with crystalloid or colloid solution (if available) at 20 ml/kg/hour as a bolus given over 15 minutes to bring the patient out of shock as quickly as possible. If the patient's condition improves, give a crystalloid/colloid infusion of 10 ml/kg/hour for one hour. Then continue with crystalloid infusion and gradually reduce to 5–7 ml/kg/hour for 1–2 hours, then to 3–5 ml/kg/hour for 2–4 hours, and then to 2–3

ml/kg/hour or less, which can be maintained for up to 2–3 ml/kg/hour or less, which can be maintained for up to 24–48 hours. If vital signs are still unstable (i.e. shock persists), review the haematocrit obtained before the first bolus. If the haematocrit was low (<40% in adult females, <45% in adult males), this indicates bleeding and the need to crossmatch and transfuse blood as soon as possible. If the haematocrit was high compared to the baseline value (if not available, use population baseline), change intravenous fluids to colloid solutions at 10–20 ml/kg/hour as a second bolus over 30 minutes to one hour. After the second bolus, reassess the patient. If the condition improves, reduce the rate to 7–10 ml/kg/hour for 1–2 hours, then change back to crystalloid solution and reduce the rate of infusion as mentioned above. If the condition is still unstable, repeat the haematocrit after the second bolus. If the haematocrit increases compared to the previous value or remains very high (>50%), continue colloid solutions at 10–20 ml/kg as a third bolus over one hour. After this dose, reduce the rate to 7–10 ml/kg/hour for 1–2 hours, then change back to crystalloid solutions and reduce the rate of infusion as mentioned above when the patient's condition improves. Further boluses of fluids may need to be given during the next 24 hours. The rate and volume of each bolus infusion should be titrated to the clinical response. Patients with severe dengue should be admitted to the high-dependency or intensive care area.

The host defense response to dengue is similar despite the tissue involved. It consists of an interactive network of simultaneously activated pathways that work in an integrated fashion to increase the host's change of survival. It is now recognized that when inflammatory signals reach the cell surface they initiate a series of events leading to activation of cytoplasmic transcription factors: DNA-binding proteins that regulate the transcription of target genes into messenger RNA. Important transcription factors associated with the host defense response include nuclear factor- κB (NF-κB). NF-IL-6, activator protein-1 (AP-1), the glucocorticoid receptor complex, and the heat shock transcription factor (HSF). Methylprednisolone as glucocorticoid influence cellular function mainly by change gene regulation through signal transmission directly to the nucleus of the cell. NF-κB is recognized as the transcription factor critical for maximal expression of multiple cytokines. The activated form of NF-κB then moves rapidly to the nucleus, initiating mRNA transcription of proinflammatory cytokines: tumor necrosis factor-α (TNF-α), interleukins (IL-1β, IL-2, IL-6), chemokines (IL-8), cell adhesion molecules (intercellular adhesion molecule-1, E-selectin), and inflammation-associated enzymes [cyclooxygenase (COX), phospholipase A2 (PLA2), inducible nitric oxide (iNOS). Cellular responses in host defense response are regulated by a complex interaction among cytokines with final effects on the surrounding microenvironment not directly induced by the initiating insult. In this regard, cytokines have concentration-dependent biologic effect. At low concentration they regulate homeostasis, and at progressively higher concentrations they mediate proportionally stronger local and then systemic respons. Methylprednisolone inhibited inflammatory cytokines secretion by influencing post translation process which cause loss of mRNA which coding cytokines. Methylprednisolone therapy is effective for DHF patient before the fifth day of fever. In DHF infection after the fifth day of fever, platelet declination did not happened. In the time of DHF diagnostic, all immunology reactions has been showed, methylprednisolone as anti inflammatory agent could inhibit over reaction of immunology. Five things which cause steroid therapy failure are that the dose which have been given did not the dose of anti-inflammatory. The type of steroid is not the strong group of steroid, the late of steroid therapy (in the time of there is shock or DSS after the fifth day of fever with platelet antibody (-), patient with gastrointestinal (GIT) bleeding, occult bleeding (hematuria and menstruation).

In this research all of patients to reseive methylprednisolone and colloid. The dose methylprednisolone 125 mg twice a day and colloid (MW 40 kDa) 500cc per day for 3 days has been given to the patient whose fulfilled the inclusion criteria. We found temperature change to normal level after intervention. Methylprednisolone and colloid therapy is effective to accelerade the temperature, IL-1β, TNF-α, and PLA-2 level declination, accelerate the endothelial cells sealing and plasma migration in dengue infective patient.

Treatment entails fluid therapy, the appropriate use of volume support crystalloid and or colloids. Colloid is the liquid which has high molecular weight and the sealing effect for the case of endothelial cells leakage. The best colloid is the one which has molecular weight in the range 100–300 kDa especially

in DSS. In this research, we used colloid with 40 kDa molecular weight in this research with the aim that it could be give in the early phase of DHF (for therapy and prevention to DSS).

According to the research of Panpanich, 2006 and Chatham, 2007, there are tendency to find the benefit of steroid in the management of DHF (relative risk less than 1, although it did not significant statistically). In this research, we found that there are significant differences between pre and post intervention using methylprednisolone (Medixon®) and colloid (Haemacell®) ($p = 0.001$ for PLA-2, $p = 0.001$ for TNF-α, and $p = 0.001$ for IL-1β, significant if $p < 0.050$).

CONCLUSION

In conclusion, methylprednisolone and colloid intervention effective for preventing the progress to severe grade of dengue infection. This is because methylprednisolone could inhibited inflammatory cytokines secretion. The benefit of colloid in dengue infection is cause by its sealing effect to face endothelial cells leakage.

RECOMMENDATION

Methylprednisolone and colloid were generic name and available in the most hospital including private and government hospital, moreover they were including in health insurance in Indonesia. Methylprednisolone and colloid present in hospital formulation and rationale use in guidance therapy for special case in infection. Average long of stay patients in hospital becomes shorter, so costs cheaper, morbidity and mortality decrease potential.

REFERENCES

Azeredo EL, Zagne SM, Santiago MA, Gouvea AS, Santana AA, Neves-Souza PC, 2001. Characterization of lymphocyte response and cytokine patterns in patients with dengue fever. *Immunobiology,* 204, p. 494–507.

Chatham WW, 2007. Glucocorticoid effects on innate and acquired immunity. Uptodate 15.1. www.uptodate.com. Diakses pada 2 Juni 2008.

Depkes RI, 2004. Tatalaksana demam berdarah dengue di Indonesia. Editor: Hadinegoro SRH, Soegijanto S, Wuryadi S, Suroso T. Edisi ketiga. Jakarta. Ditjen P2M Depkes RI, h. 25–55.

Funahara Y, Ogawa K, Fujita N, et al, 1987. Three possible triggers to induce thrombositopenia in Dengue virus infection. *Southeast Asia J. Trop. Med. Pub. Hlth* 18, p. 351–355.

Gubler DJ, 1998. Dengue and dengue hemorrhagic fever. *Clin. Microbiol. Rev.* 11, p. 480–496.

Guglani L, Kabra SK, 2005. T Cell Immunopathogenesis of Dengue Virus Infection. *Dengue Bulletin* 29, p. 58–69.

Hadinegoro SR, 1996. Telaah endotoksemia pada perjalanan penyakit demam berdarah dengue: perhatian khusus pada syok, produksi TNF alfa, interleukin-6, dan sebagai faktor prediktor demam berdarah dengue berat. *Disertasi*, Universitas Indonesia press Jakarta.

Halstead SB, 1992. Pathogenesis of dengue: challenge to molecular biology. *Science* 45, p. 292–298.

Kautner I, Robinson MJ, Kubnle U, 1997. Dengue virus infection: Epidemiology, pathogenesis, clinical presentation, diagnosis and prevention. *J pediatrics* 131, p. 516–524.

Koraka P, Suharti C, Setiati TE, Mairuhu AT, Van Gorp E, Hack CE et al, 2001. Kinetics of dengue vurus-spesific immunoglobulin classes correlate with clinical outcome of infection. *J Clin Microbiol* 39, p. 4332–4338.

Krishnamurti C, Peat RA, Cutting MA, Rothwell SW, 2002. Platelet-adhesion to dengue-2 virus infected endothelial cells. *Am J Trop Med Hyg* 66, p. 435–441.

Kusriastuti R, Sutomo S, 2005. Evolution of Dengue Prevention and Control Programme in Indonesia. *Dengue Bulletin* 29, p. 1–7.

Kurane I, 2002. Antibody dependent enhancement of infection. dengue virus immunology and immunopathology. Available at: www.science.mcmaster.ca/biology Assessed on December 9th, 2006.

Lei HY, Yeh TM, Liu HS, Lin YS, Chen SH, Liu CC, 2001. Immunopathogenesis of dengue virus infection. *J Biomed Sci* 8, p. 377–388.

Livingstone, 1995. Modulation of the function of dengue virus specific human CD8+ cytotoxic T cell clone by IL-2, IL-6 and IFN gamma. *Immunol. Invest.* 24, p. 619–629.

Meduri, 2000. rationale for Glucocorticoid Treatment in Septic Shock and Unresolving ARDS. In: Multiple Organ Failure Pathophysiology, Prevention, and Therapy. Editors: Baue AE, Faist E, Fry DE. New York, p. 514–521.

Nasronudin, 2005. Fluid therapy in dengue hemorrhagic fever: Role of HES. Editor: Hermawan G, Pramana Y, Prasetyo DH . Dalam: Kumpulan makalah lengkap : Raih ilmu kedokteran terkini rengkuh Indonesia sehat 2010. Kongres Nasional PETRI XI, PERPARI VII, PKWI VIII., PIT II PAPDI cab. Surakarta. Surakarta, h. 15–29.

Nasronudin, 2007. Imunopatofisiologi Infeksi Virus Dengue. In: Penyakit Infeksi di Indonesia. Ed: Nasronudin, Usman Hadi, Vitanata, Erwin AT, Bramantono, Suharto, Eddy Soewandojo, h. 40–56.

Noisakran S, Perng GC, 2008. Alternate Hypothesis on the Pathogenesis of Dengue Hemorrhagic Fever (DHF)/Dengue Shock Syndrome (DSS) in Dengue Virus Infection. Exp Biol Med 233, 401.

Oppenheim JJ, Ruscetti FW, Faltynek C, 1991. Cytokines. Stites DP, Terr AI eds. In: Basic and Clinical Immunology. 7th Edition . London. Prentice Hall International Inc., p. 78–99.

Wang S, He R, Patarapotikul J, Innis BL, Anderson R, 1995. Antibody-enhanced binding of dengue-2 virus to human platelets. Virology 213, p. 254–257.

WHO, 1997. Dengue haemorrhagic fever: diagnosis, treatment and control. Geneva, p. 17–22.

WHO, 2009. Clinical Management and Delivery of Clinical Services. In: Dengue Guidelines for Diagnosis, Treatment, Prevention and Control. New Edition, p. 25–39.

THE INTERVENSION OF STANDARD AND PERSUASIVE MESSAGES IN HEALTH PROMOTION EDUCATION TOWARD PRACTICES OF PREGNANT WOMEN IN THE DISTRICT OF BUOL

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ABSTRACT

Background: *This research was about the maternal health practice effect of the persuasive messages in health promotion. The hypothesis was that the persuasive messages in health promotion enhance the maternal health promotion.* ***Methods:*** *The research was experimented with the pre-posttest control group design. The population was all pregnant women of the third trimester in sub district of Momunu and Lipunoto. Samples were selected by cluster sampling of 28 women. The data was analyzed by t- test and Wilcoxon test with degree of significance 95%.* ***Results:*** *Showed that both models in the maternal health promotion are significant in increasing the knowledge, attitude and practice from the pre and posttest ($p < 0.05$). The comparison of influences both types of intervension were not different ($p > 0.05$), but it suggests that the additional persuasive messages in health counseling are needed to inform importances of communication messages strategy and comprehensive message design in every health promotion activity.*

Key words: *persuasive, maternal health practice, health promotion standard*

ABSTRAK

Penelitian ini adalah tentang pengaruh praktik kesehatan ibu dari pesan persuasif dalam promosi kesehatan. hipotesis adalah bahwa pesan-pesan persuasif dalam promosi kesehatan meningkatkan promosi kesehatan ibu. Penelitian ini bereksperimen dengan rancangan pra-postes kelompok. Populasi adalah semua ibu hamil trimester ketiga di Kecamatan Momunu dan Lipunoto. Sampel dipilih oleh cluster sampling dari 28 perempuan. Data dianalisis dengan t-test dan uji Wilcoxon dengan tingkat kepercayaan 95%. Hasil penelitian menunjukkan bahwa kedua model dalam promosi kesehatan ibu adalah signifikan dalam meningkatkan pengetahuan, sikap dan praktik dari sebelum dan sesudah ($p < 0,05$). Perbandingan pengaruh kedua jenis intervensi tidak berbeda ($p > 0,05$), namun menunjukkan bahwa pesan persuasif tambahan dalam penyuluhan kesehatan yang dibutuhkan untuk menginformasikan pentingnya strategi komunikasi pesan dan desain pesan yang komprehensif dalam setiap kegiatan promosi kesehatan.

Kata kunci: *persuasif, praktik eksehatan ibu, standar promosi kesehatan*

Submit: 13 Mei 2010, Review 1: 20 Mei 2010, Review 2: 20 Mei 2010, Eligible articles: 28 Mei 2010

INTRODUCTION

The maternal mortality rate in Indonesia is considered very high. The Indonesia demographyc survey of health year 1995 showed the maternal mortality rate was 373 per 100,000 live births, whereas the survey year 1997 showed that the rate was 334 per 100,000 live births. And result Indonesia demography survey year 2002–2003 showed that the maternal mortality rate was 307 per 100,000 per live births. The maternal mortality rate in Central Sulawesi – 384 per 100,000 live births - was higher than that of the national. Area with the highest maternal mortality rate in Central Sulawesi is district of Buol is 693 maternal deaths per 100,000 live births. Though, the maternal mortality rate tends to decrease every year, it seems that it is quite difficult to achieve the national target 125 maternal deaths per 100,000 live births by 2010. According to Central Sulawesi Provincial Health office,

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